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RETINAL VEIN OCCLUSION

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APC RESISTANCE IN PORTUGUESE CAUCASIANS PATIENTS WITH RETINAL VEIN OCCLUSION.

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A poor anticoagulant response to activated protein C (APC resistance) induced by the Arg/Gln506 mutation of the Factor V gene is, in several countries, the most common genetic defect associated with thrombophilic disorders.
Frequencies of APC resistance between 20-50% have been observed in patients with deep venous thrombosis, suggesting the importance of the Protein C system activity. Also, in other thrombotic situations as myocardial infarction or thrombosis of cerebral vessels, APC resistance has been occasionally referred.
In a previous study, we have found a prevalence of 1.5% (95%IC 0.31 - 4.32) for APC resistance, in the general portuguese Caucasian population.
*Purpose:*The aim of the present study is to evaluate the prevalence of APC resistance in one group of patients with retinal vein occlusion (RVO) where the contribution of the Protein C system is controversial and the role of APC resistance not yet established.
Methods: In all patients, APC ratio (Chromogenix COATEST APC resistance kit in a bioMérieux Hemolab coagulometer) and Factor V gene mutation (PCR and MnlI digestion) were determined according to the recommended protocols. The cut off point of 2.5 for APC ratio achieves the best agreement between phenotypic and genotypic data.
We have studied 52 consecutive unrelated patients with an history of RVO defined by Hayreh's criteria.
Results: None of the observed patients carried the mutation and the APC ratio mean±SD was of 4.12 ± 0.83 . However, one patient had an APC ratio of 1,7.
Conclusions: The frequencies found in the studied group are identical to those of the general population. In this way APC resistance, does not play a major role in the possible thrombophilic component in RVO.

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Title : Local Intra-arterial fibrinolysis for central venous occlusion

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Purpose : To study the effects of urokinase perfusion in the ophthalmic artery in recent central retinal venous occlusion (CRVO)

Methods: Eight patients with CRVO were treated. All cases were non-ischaemic forms of venous occlusion, with severe visual loss at onset, and/or masked delayed retinal circulation time, and/or CRVO in the second eye, or a previous episode in the same eye. The symptoms lasted from 12 h to two weeks at presentation.
A microcatheter was introduced through the femoral artery and placed at the origin of the ophthalmic artery. Then 250 000 IU of urokinase was injected at a speed of 1 ml/min.
For all patients, fibrinolysis was followed by heparinization for 4 to 6 weeks. Follow up ranged from 6 to 18 months.

Results: Vision improved at the end of fibrinolysis in 4 of the 8 patients. Their retinal circulation time improved markedly after treatment, by one day. These 4 patients exhibited complete normalization of the fundus within 2 to 4 weeks. The other patients did not exhibit any noticeable improvement or their vision. No complication occurred.

Conclusion: This preliminary study shows that in come cases of CRVO, local intra-arterial fibrinolysis may result in an immediate striking improvement of vision and circulation times.

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RETINAL GANGLION CELL SURVIVAL AFTER TRANSIENT ISCHEMIA OF THE RETINA: EFFECTS OF INTRAOCULAR INJECTION OF BDNF.

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Purpose To investigate the effects of different transient periods of ischemia and intraocular administration of BDNF on retinal ganglion cell survival. ***Methods*** In adult rats, we have applied Fluoro-gold (FG) to both superior colliculi, the main retino-recipient target nuclei in the brain. One week later, when most retinal ganglion cells (RGCs) have incorporated the dye into their cytoplasm, the left retina of several groups of animals underwent transient periods of complete ischemia of 30, 45, 60, 75, 90, 105 or 120 minutes duration by raising intraocular pressure of the eye above systolic levels. The rats were perfused through the heart 5, 7, 15 or 30 days later, their retinas prepared as flattened whole-mounts and examined under fluorescence microscopy for FG-labelled RGCs. Mean densities of surviving (FG-labelled) RGCs were estimated by counting these cells in twelve standard areas of the retina according to previously described methods (Villegas-Pérez et al., J. Neurobiol. 1993, 24:23). In additional groups of animals, right after a transient ischemic period of 90 minutes, the eye was punctured or injected with 5 µl of saline containing 0,1% BSA alone or with 5 µg of BDNF, and their retinas processed as above 7 days later.
Results In the groups of rats analyzed at 5, 7, 15 and 30 days we found that: i) Periods of ischemia below 45 minutes did not cause significant decreases of RGC densities; ii) After periods of ischemia of 60 minutes, RGC survival was 67%, 63%, 51% and 45% respectively. After periods of ischemia of 90 minutes, RGC survival was 52%, 43%, 16% and 4% respectively. iii) After periods of ischemia of 105 and 120 minutes, only a small population of RGCs survived 15 and 30 days after ischaemia. iv) The groups of retinas treated with BDNF, vehicle or sham injection showed moderate increases in FG-labelled RGC densities when compared to untreated retinas.
Conclusions In the adult rat retina: i) Transient periods of ischemia of 30 and 45 minutes do not induce RGC death; ii) Longer periods of ischemia induce the death of a proportion of RGCs that increases with the duration of the period of ischemia; iii) Periods of transient ischemia above 90 minutes induce the death of approximately 95% of the RGC population; iv) RGC death can be observed as soon as 5 days after transient ischemia, but continues during the period of this study (up to 30 days), and; v) Intraocular injection neuroprotects a small population of RGCs. Supported by FIS 95/1720 & CARM-PIB94-15 grants. Regeneron Pharmace. Inc., provided BDNF.

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FREE RADICALS ARE PRODUCED IN THE RETINA DURING ISCHEMIA - IN VIVO ELECTRON SPIN RESONANCE STUDY IN THE RABBIT

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Purpose : Pharmacological experiments have suggested that ocular ischemia induced by high intraocular pressure (HIOP) in the rabbit, provoked an oxidative stress responsible for functional alteration of the retina. However the nature of the oxidant chemical species and their mode of generation were not elucidated. The aim of the present studies was to characterize the oxygen-derived free radicals produced during and/or after the hyperpressure period, by electron spin resonance (ESR) analysis.

Method : HIOP was induced in the rabbit eye by perfusing the anterior chamber with saline at 100 mmHg for 45 min. A microdialysis probe placed in contact to the retina was perfused with buffer containing the spin trap DEPMPO. Continuous flow of retinal superfusate was analysed in an ESR spectrometer. Ascorbate was also measured in the retinal tissue after ischemia or ischemia/reperfusion. In addition flash electroretinograms were recorded to determine the functional consequences of HIOP and free radical generation.

Results : Hydroxyl radicals were generated during the HIOP period and the oxidative stress was not increased at reperfusion as assessed by ESR and ascorbate measurements. Functional protection afforded by free radical scavengers (superoxide dismutase + catalase, tempo + catalase and dimethylthiourea) against HIOP-induced ERG alteration confirmed these observations.

Conclusion : Our results show, from the first time, that hydroxyl radicals are produced in the retinal during the ischemic period itself and that oxidative stress is responsible for ascorbate fall and ERG extinction.